III.7 Bioindicator Species for Evaluating Potential Effects of Pesticides on Threatened and Endangered Wildlife

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Monitoring pesticide applications for possible effects on wildlife is an integral part of pesticide registration and regulation and of a successful grasshopper integrated pest management (GHIPM) system. During grasshopper outbreaks, U.S. Department of Agriculture cooperative grasshopper control programs have treated as much as 13.1 million acres (5.3 million ha) of rangeland in a single season (U.S. Department of Agriculture, Animal and Plant Health Inspection Service 1987).

Large numbers of insectivorous birds may inhabit, or congregate in, areas where these insecticide applications are made. One grasshopper egg bed found in Otero County, CO, encompassing 2 acres (0.8 ha), was populated by “about 200 western horned larks and lark buntings,” which were seen feeding heavily on the grasshopper nymphs (Wakeland 1958). An effective GHIPM program should retain the natural controls on grasshoppers and not disrupt the rangeland ecosystem, including threatened and endangered species.

Wiens and Dyer (1975) reported breeding-season bird densities averaging approximately 0.8 to 1.3 birds/acre (1.9 to 3.3 birds/ha) on rangeland. Johnson et al. (1980) summarized avian densities for grassland–sagebrush habitats as averaging 1.2 to 5.0 breeding birds/ha. Therefore, large numbers of birds and other wild vertebrates can be exposed to a chemical during a single pesticide application (McEwen 1987). In areas not monitored during an application, mortality, and particularly sublethal effects, caused by pesticides can be overlooked because mortality “usually affects only part of the fauna, is scattered in space and time, and generally occurs where there is no biologist to record it” (Stickel 1975).

Toxicity evaluation has employed the use of white rat species in a laboratory setting utilizing test animals that are common species, easily bred, maintained, and handled. Controlled tests are pertinent for determining baseline data and comparing relative toxicity of chemicals. However, to understand pesticide effects in the natural environment, all the intricate interactions of cover, weather, food, exposure routes, and animal behavior, must be considered. Toxicity tests in the laboratory can only predict ecotoxicity in the field setting within broad limits.

An intermediate step between laboratory and field investigations is the use of caged or penned vertebrates located within an application block as used by Kreitzer and Spann (1968). However, it was found that the cage-in-field method resulted in less exposure to the pesticide than free-ranging wildlife received and actually protected the experimental animals from possible predation related to sublethal effects (Heinz et al. 1979).

Sublethal effects can be observed in the controlled environment of laboratory investigations, and researchers often surmise that “a sublethal effect seen in the laboratory would also occur in the field and that this effect would result in mortality or reproductive problems” (Heinz 1989). These effects can also be misleading or overlooked. For example, Grue et al. (1982) found that free-living starlings differed from captive birds by losing weight after dosing with dicrotophos, an organophosphate (OP) insecticide. Field investigations are a necessary step in evaluating the overall effects of large-scale pesticide applications.

It has been recognized that data on effects of OP’s and other classes of pesticides are incomplete (Grue et al. 1983, Kirk et al. 1996). The Avian Effects Dialogue Group (1994) set forth some recommendations for more effective techniques in gathering data. Several issues of concern were studies on focal avian species, study sites, carcass searching, population changes, modeling, use of radio telemetry, and dissemination of information.

Species of critical concern are usually unavailable for any hands-on laboratory or field toxicity studies, thus making the need for surrogate species a necessity. Lower and Kendall (1990) suggested some criteria for selecting a sentinel species (one in which effects may be interpreted as indicators of similar disturbances in other species) when evaluating synthetic compounds, such as pesticides in the field. This approach has several limitations.

For example, can the toxicity of a chemical to a chicken, duck, or quail predict toxic effects on a falcon or eagle? How do the differences in a species’ physiology, food, habitats, and ecology affect the animal’s exposure and reaction to the chemical? When threatened or endangered (T and E) species may be at risk, they of course, cannot be collected for chemical analysis, pathology examination, or food-habits study. Thus, the next best approach is to estimate potential effects on T and E species by study of closely related sentinel species.

The American kestrel *(Falco sparverius)* has been shown to be more sensitive to anticholinesterase insecticides than other avian species (such as quail and ducks) used to establish toxicity (Rattner and Franson 1984, Wiemeyer and Sparling 1991). Consequently, the kestrel is a conservative bioindicator of possible effects on the related peregrine falcon *(Falco peregrinus).*

Our environmental monitoring team's studies have utilized the American kestrel and killdeer *(Charadrius vociferus),* as surrogates for other Falconiformes and Charadriidae, such as the peregrine falcon and mountain plover *(Charadrius montanus),* respectively. Kestrels and killdeer are representative of their genera, are widely distributed, and are found in much greater numbers than their endangered relatives.

The American and European kestrels have been utilized in toxicology studies for many years (Wiemeyer and Lincer 1987). Studies of the American kestrel, the smallest and most abundant falcon throughout North America, have progressed from laboratory toxicity tests to field ecotoxicology investigations over the past 20 years. Since kestrels are commonly present on rangelands where grasshopper outbreaks occur, they are excellent subjects for examining direct and indirect effects of control programs. Kestrel use of nest boxes (fig. III.7–1) and tolerance of disturbance and observers makes it possible to investigate all stages of their life cycle. Henny et al. (1983) examined productivity of free-ranging kestrels using nest boxes beginning in 1978 for investigating the adverse effects of the pesticide heptachlor in Oregon's Columbia River Basin.

Figure III.7–1—Kestrel nest box used on rangeland. Access to the eggs and nestlings is through a hinged side of the box. Field crews can check nests periodically to determine egg hatchability, growth measurements, and survival of young, and to affix leg bands and attach transmitters. (Photo by L. C. McEwen of Colorado State University; reproduced by permission.)

On rangelands, population densities of American kestrels may be restricted by the lack of natural tree cavities for nesting sites. Investigation of pesticide effects could be difficult to document because of small sample sizes of kestrels, but nesting populations can be increased by adding artificial nest box structures. Frocke (1983) summarized the use of nest boxes in avian management and research; cavity-nesting species have exhibited a readiness to use, and possibly a preference for, nest boxes over natural cavities. Kestrels are very adaptable and will easily accept the use of human-made nest boxes.

Kestrels favor open-space sites for hunting, so establishing new nest sites in these open areas for experimental purposes can be effective. Although Loftin (1992) found in Florida that nest boxes placed in pastures or areas away from known kestrel use were ineffective in increasing American kestrel populations, we did not find this to be true. We had >50 percent use of all nest boxes in six different geographic locations from Colorado to Alaska. However, in some areas, it took 2–3 years to reach maximum use of boxes. (Plans and directions for construction and placement of nest boxes are given in chapter I.11 of this Handbook.)

Seven years of production data have been compiled on nesting American kestrels during the Grasshopper Integrated Pest Management (GHIPM) Project. Approximately 560 nest boxes were in place by the sixth year among 6 locations: the 2 GHIPM demonstration areas in Idaho and North Dakota, Alaska, Wyoming, and 2 parts of Colorado—the northwestern section and in the Front Range (fig. III.7–2). Data on clutch size, hatchability, and numbers of nestlings fledged were collected annually (table III.7–1).

Productivity is presented as baseline data for each location and compared between years. Mean clutch sizes did not vary among locations, but yearly differences were observed ($P < 0.05$). Alaskan kestrels surpassed birds

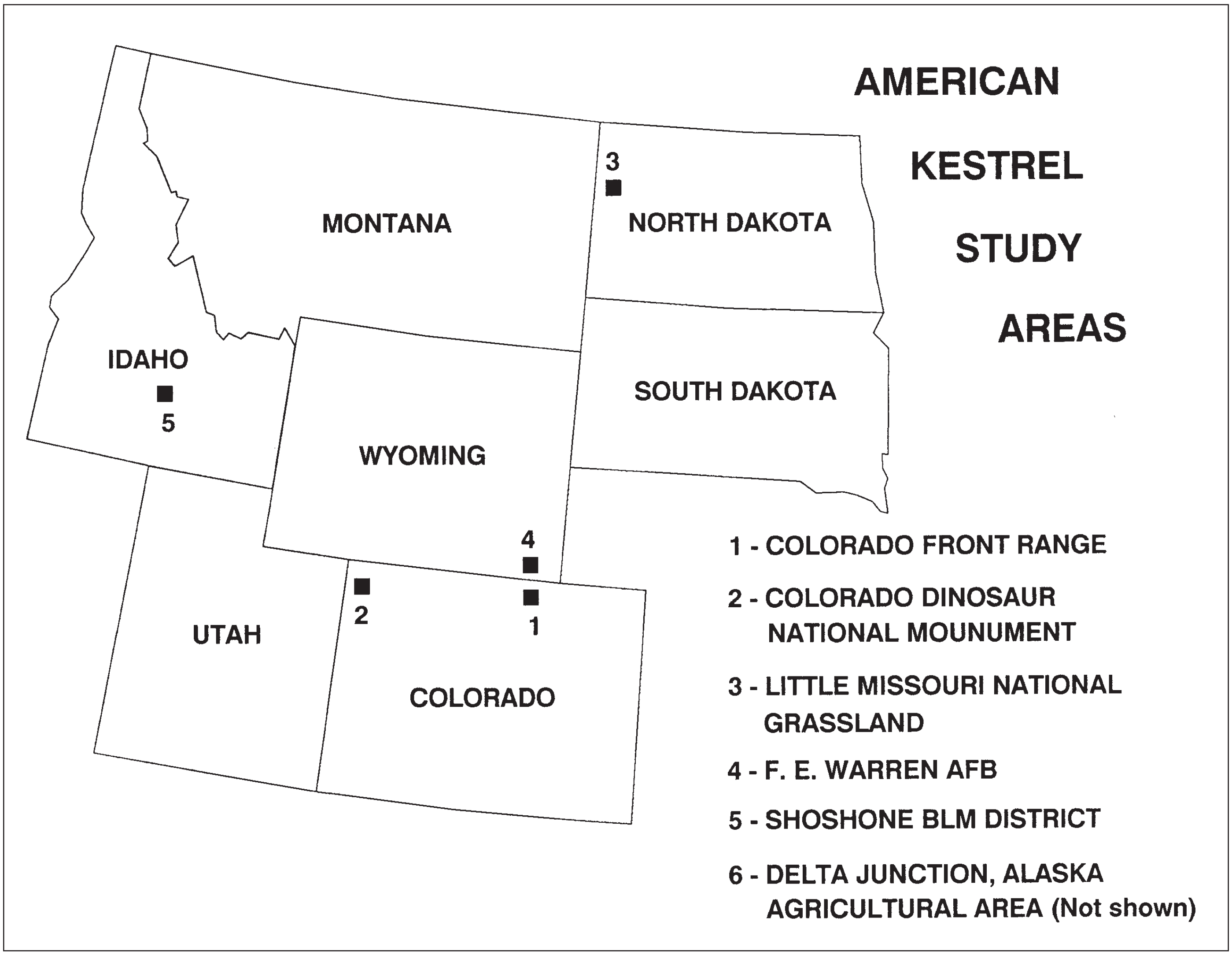

Figure III.7–2—Locations of kestrel study areas where >500 nest boxes have been placed (total of all areas). Key: 1 = Colorado, Front Range; 2 = Colorado, Dinosaur National Monument; 3 = Little Missouri National Grasslands; 4 = F. E. Warren Air Force Base; 5 = Bureau of Land Management's Shoshone District. (A sixth location, an agricultural area in Delta Junction, AK, is not shown.)

Table III.7–1—Variation in nesting productivity of American kestrels in the GHIPM demonstration areas and other treatment and reference areas during 1988–94

Location and years	Mean no. of nests/yr	% of nests hatched[1]	% of nests fledged[2]	Mean no. fledged per nest attempt
Alaska				
1990–93	33	85–97	82–97	3.5–4.3
Colorado, Front Range				
1988–94	26	61–88	55–81	2.0–2.9
Colorado, northwestern				
1988–94	24	81–89	79–84	2.9–3.1
Idaho				
1988–93	62	60–90	48–81	1.8–3.5
North Dakota				
1988–94	83	58–88	50–70	1.5–3.0
Wyoming				
1989–94	12	31–100	19–100	0.6–3.8

[1] Hatched nest: ≥ 1 egg hatched.
[2] Fledged nest: ≥ 1 young fledged.

from all other areas sampled in mean number of eggs hatched and young fledged in 1990 through 1993, but the differences were not statistically significant ($P > 0.05$).

Lower kestrel productivity in Idaho and North Dakota coincided with drought years and with the one extreme high-precipitation year in the Dakotas but otherwise was similar for most years (table III.7–1). The results illustrate the variability in kestrel nesting success due to natural factors and emphasize the importance of having concurrent untreated nest boxes for observation when investigating possible pesticide effects on nests in sprayed areas. Comparison of comparable untreated nests with sprayed nests over the same time period, is necessary to differentiate effects of weather, predation on nestlings by great horned owls *(Bubo virginianus),* and other natural factors from pesticide treatment effects.

In 1990–94, a limited number of nest boxes in several locations, excepting Idaho, were used to study sublethal effects on kestrel nestlings and fledglings of (1) *Beauveria bassiana,* a fungus bioinsecticide; (2) carbaryl, a carbamate (sprays and bran-bait treatments); (3) malathion, an organophosphate; and (4) diflubenzuron (Dimilin®), an insect growth regulator. These results are presented in separate sections.

Field Applications

A carbaryl bran-bait treatment was examined at the Delta Agricultural Project in Alaska where five kestrel nest sites with heavy grasshopper infestation were selected for study of the effects of carbaryl bait. At the time of application, nestlings were approximately 18–22 days of age. Three of these nests had 2 percent carbaryl bran-bait applied at approximately 2.2 lb/acre on 40 acres (16.2 ha) adjacent to the nest box entrances, and 2 nests were left untreated. No adverse effect was noted on the treated nests, and all kestrel nestlings fledged normally. It was also found that numbers of breeding birds in North Dakota on line transects before and after application did not differ when controlling grasshoppers with carbaryl bait (George et al. 1992).

Possible effects on killdeer from spray applications of two formulations of Sevin® 4-Oil (20 or 16 fl oz/acre, with each containing 4 fl oz of diesel oil; active ingredient [AI] of carbaryl was 8 and 6.4 fl oz/acre or 0.56 and 0.45 kg/ha, respectively) were investigated in North Dakota during 1992. Brain AChE activities were monitored at 2, 8, and 21 days after applications and found not to differ from normal (Fair et al. 1995). Whole body carbaryl residues were low (averaging <0.1 to 1.4 p/m [parts per million]) but significantly *(P* < 0.05) greater for birds collected from the sprayed areas compared to birds from unsprayed surrounding locations. No toxic signs were observed in any killdeer. On the treated areas, birds captured invertebrate prey at rates significantly higher than on reference areas at 2 and 8 days after spraying (Fair 1993) presumably due to the availability of dying insects.

Acute Oral Dosing Treatments and Procedures

Growth, nestling and fledgling survivability, and postfledging movements of young wild kestrels were measured in the field after exposure to an acute sublethal oral dose of one of the following standard or experimental IPM materials: *Beauveria bassiana*, diflubenzuron, carbaryl, malathion, or their formulation carriers (diesel or corn oil). A minimum of four young per brood were used in these studies. The remaining nestling(s), if any, in each box served to maintain a normal brood size and provided an untreated comparison to the dosed birds. Their ages varied from 8 to 16 days when nestlings were randomly selected and given a single dose of one of the following: corn oil, pesticide formulation, the petroleum-based oil used in the formulation (carrier oil or #2 diesel fuel), or the technical material. Behavior and growth data were collected every 4 days following dosing.

Surviving test nestlings were fitted with transmitters at 26–31 days of age (fig. III.7–3). After fledging, all birds were located daily or every other day until transmitters failed or young moved too far from the nest box area to be located.

Beauveria bassiana Sublethal Test

This investigation was conducted in the short-grass prairies of north-central Colorado during 1992. Thirteen nest boxes containing 55 young were tested (table III.7–2). Two of the nests were given challenge dosages of 5 µL

Figure III.7–3—Young kestrel with small transmitter attached for the study of postfledging behavior, movements, and survival. (Photo by B. E. Petersen of Colorado State University; reproduced by permission.)

Table III.7–2—Survival of American kestrel nestlings dosed with *Beauveria bassiana* formulation, carrier oil, corn oil, or untreated in north-central Colorado, May–August 1992

	Beauveria formulation[1]	Carrier oil[2]	Corn oil[2]	Untreated control
No. nestlings dosed	14	13	13	15
No. nestlings survived	11	12	13	15
No. fledglings with radios	11	12	13	2
No. fledglings survived	10	10	12	2

[1] Contains formulation oil and *Beauveria bassiana* spores. Dosage was based on 500,000 spores/µL and 1 µL/g of body weight.

[2] Dosages based on 1 µL/g of body weight.

(microliters)/gram of body weight for the formulation and carrier oil; for the main test, broods were dosed at 1 µL/gram of body weight. No statistical significance was detected in either growth rates or behavior data among treated and untreated groups ($P > 0.05$). Transmitters were attached to 38 kestrels. Data were collected on survival and movements of 28 of those birds (10 radio attachments failed). No detectable differences in survival or movements were found among treated and untreated kestrels.

Seven treated fledglings, ages 31–42 days, were collected for examination. Two additional fledglings were found dead and also the remains of one eaten by predators. Necropsies were performed on all collected birds at the Colorado Veterinary Teaching Hospital; no visible gross pathology was detected.

Diflubenzuron Sublethal Test

This investigation was conducted in north-central Colorado during 1993–94. Forty nest boxes containing 170 young were used (table III.7-3). Two of the nests were given preliminary challenge dosages of 64 mg/kg of body weight of technical diflubenzuron (Dimilin) to estimate toxicity, if any. (In English measure, this is the equivalent of 0.0009 oz diflubenzuron per pound of body weight). All following dosages will be given in metric units as used in toxicology. Kestrel broods in the main study were dosed at 10.2 mg/kg.

No statistical differences were detected in nestling growth rates, behavior data, or survival among treated and untreated birds ($P > 0.05$). Although no differences were found in nestlings, possible effects on fledgling survival were seen the first year. Transmitters were attached to 42 fledgling kestrels. During 1993 approximately half the fledgling kestrels dosed with diflubenzuron formulation died or were lost, warranting a second year of research. In 1994, however, more than 70 percent of the 43 kestrels fitted with transmitters survived, and no differences were observed between treated and control fledglings.

Several treated fledglings, ages 27 to 45 days, were found dead due to predation or other causes. Necropsies were performed on all the dead birds, and no gross pathology was detected.

Carbaryl Sublethal Test

American kestrel nestlings in nest boxes on the North Dakota GHIPM demonstration area were administered sublethal acute oral doses of Sevin 4-Oil formulation in 1992 to determine effects on growth and postfledging survival. Two 10-day-old nestlings were given 200 mg/kg body weight of Sevin 4-Oil (40.5 percent carbaryl or 81 mg/kg AI) to establish a lethal dosage. Brain acetylcholinesterase (AChE) activity was depressed 80 percent at death in 27–35 minutes. Four additional nestlings all survived Sevin 4-Oil dosages of 30–100 mg/kg.

Table III.7–3—American kestrel nestling and fledgling survival after dosing with technical or formulation diflubenzuron, diesel oil #2, corn oil, or untreated in north-central Colorado during 1993–94

	Diflubenzuron Technical	Diflubenzuron Formulation	Diesel oil #2	Corn oil	No treatment
No. nestlings dosed	[1]40	40	40	39	11
No. nestlings survived	32	33	34	32	10
No. fledglings with radios	25	27	27	6	—
No. fledglings survived	22	19	21	3	—

[1] One bird dosed with technical diflubenzuron was collected prior to radio transmitter fitting.

Sublethal dosages then were given to 32 nestlings (8 to 14 days old). Sixteen were dosed at 15 mg/kg and 16 at 30 mg/kg with Sevin 4-Oil. Sixteen additional nestlings were given corn oil at 2 µL/g of body weight as untreated controls subjected to the same handling procedures. Blood samples were collected from the nestlings for analysis of plasma cholinesterase activity at 1 hour, 24 hours, and 7 to 14 days after dosing. Radios were placed on 30 of the nestlings for study of postfledging movements and survival. Twenty-one of the nestlings and fledglings were collected at 10 to 38 days after treatment for brain AChE activity measurements, carcass residue analysis, and necropsy. Carbaryl residues were no longer detectable in the carcasses, but three had 0.08–0.15 p/m in their gastrointestinal tracts (analyzed separately). No gross pathology was found.

None of the 21 birds had significant inhibition of brain AChE activity or any signs of gross pathology. The lack of brain AChE inhibition was not unexpected because of the sublethal dosage levels and the rapid reversibility of carbaryl inhibition. Blood plasma samples showed mild AChE inhibition at 1 hour after treatment (averages = 4 percent at 15 mg/kg and 12 percent at 30 mg/kg). Recovery from the low degree of plasma AChE inhibition was evident in all carbaryl-dosed nestlings by 24 hours after treatment.

Malathion Sublethal Test

American kestrel nestlings in North Dakota were administered sublethal acute oral malathion dosages in 1993 and 1994. To establish the sublethal treatment dosages, it was first necessary to determine the acute oral lethal levels by conducting preliminary range-finding toxicity tests. Based on reported malathion toxicity to other avian species, dosages ranging from 49 to 500 mg/kg were administered to seven nestlings, and all dosages were found to be lethal. In further tests, it was determined that lethal malathion dosages began at 20 to 40 mg/kg (Taira 1994). These results indicated that young kestrels are much more sensitive to malathion toxicity than many other bird species for which LD_{50}'s (lethal dose to 50 percent of the birds) range from >100 to >400 mg/kg (Smith 1987). Part of this sensitivity may be age related, but scientists do not know the acute oral LD_{50} of malathion for adult American kestrels.

Young birds in 17 nest boxes were given malathion at 1 of 2 dosage levels: 5 or 20 mg/kg. An equal number were given corn oil or left untreated. Posttreatment blood samples were taken for plasma AChE and butyrylcholinesterase (BChE) assay from each bird at 1 hour, 24 hours, and between 7 and 14 days after treatment. At the 20 mg/kg dosage, both AChE and BChE were severely inhibited (77.1 and 71.6 percent respectively) at 1 hour posttreatment (table III.7-4). AChE activity was still inhibited 60.3 percent at 24 hours. BChE recovered more quickly, showing 21.9 percent inhibition at 24 hours. Nestlings dosed with 5 mg/kg were not as strongly affected but had plasma AChE inhibition of 45.4 percent and BChE inhibition of 60.8 percent at 1 hour. These results support the conclusion from the range-finding tests that young kestrels are more sensitive to malathion than many other avian species (Taira 1994).

Nestlings that were casualties in the malathion range-finding tests were analyzed for carcass residue concentrations. Whole-carcass residues ranged from 0.38 p/m in the lowest-dosed bird (49 mg/kg) to 46.5 p/m in the highest-dosed nestling (500 mg/kg). Gastrointestinal tracts (including contents) were analyzed separately, and residues varied from 12.1 p/m to 4,860 p/m corresponding to dosage levels. Only 6 of the sublethally dosed nestlings/fledglings were recovered for analysis. Residues were not detectable except in one carcass, which contained 0.21 p/m of malathion.

Summary and Conclusions

Field studies of bioindicator species are a useful approach for estimating potential ecotoxicological effects of pest control operations on threatened or endangered (T and E) species or other wildlife species of special concern. Species selected as bioindicators should be widely distributed and relatively abundant in the habitat types subjected to pest controls. Species closely related to T and E species also may be considered “surrogates” for those species and for others of concern.

In our environmental monitoring studies, we have investigated effects on American kestrels as bioindicators for peregrine falcons (and other small raptors) and effects on killdeer as bioindicators for mountain plovers. Our data on total bird populations in treated and untreated rangeland sites also could be examined in retrospect if questions arise concerning other species such as long-billed curlews, burrowing owls, ferruginous hawks, loggerhead shrikes, or rare species of sparrows.

From our GHIPM work, these two conclusions can be drawn:

(1) Young kestrels are more vulnerable to toxicity of malathion and anticholinesterase pesticides than many other avian species. Therefore, nonspray buffer zones

Table III.7–4—Mean percentage of plasma cholinesterase (ChE) activity in malathion-dosed kestrel nestlings compared to control ChE activity

	Dosages					
	5 mg/kg			20 mg/kg		
Posttreatment collection time	ChE	Total AChE[1]	BChE[2]	ChE	Total AChE	BChE
1 hour	51.1	54.6	39.2	24.2	22.9	28.4
24 hours	74.8	73.8	80.5	46.4	39.7	78.1
7 days	94.0	94.5	91.6	89.0	86.9	101.8
14 days	98.3	100.8	88.2	94.6	97.0	84.7

[1] Acetylcholinesterase.

[2] Butyrylcholinesterase.

around active nests of the closely related peregrine falcon should always be observed when liquid pesticide formulations are applied. However, bait formulations of IPM chemicals and biologicals are safe and pose no significant hazard even if used in the immediate vicinity of the nests. Acute dosages of diflubenzuron or *Beauveria bassiana* formulations indicate very low direct toxicity to young kestrels. These materials would have no direct effects on nontarget terrestrial wildlife but might reduce the insect food base in some cases. These findings should also apply to other nesting raptors on rangeland.

(2) Studies of Sevin 4-Oil grasshopper sprays (16 or 20 fl oz/acre) indicated little or no effect on killdeer (Fair et al. 1995). Cholinesterase activity was not significantly inhibited, whole-body carbaryl residues were low (<0.1 to 1.4 p/m), and food-habits studies showed that the birds maintained adequate diets. No gross pathology was found on necropsy of the killdeer. Whole body lipids were measured as an indicator of body condition and did not differ between killdeer from sprayed and unsprayed sites.

These results indicate that Sevin 4-Oil applications at 20 fl oz/acre (0.56 kg/ha carbaryl AI) or lower pose little hazard to the closely related mountain plover, a Category 1 species that may be listed in the future as endangered. However, areas known to be in the immediate vicinity of mountain plover nests should be excluded from spray applications because of the variation in individual bird response to synthetic chemical compounds. Bait formulations would be the least hazardous method of grasshopper control in mountain plover habitat.

References Cited

Avian Effects Dialogue Group. 1994. Final report of the AEDG, 1988-1993. Washington, DC: RESOLVE, Center for Environmental Dispute Resolution. 156 p.

Fair, J. M. 1993. The effects of carbaryl grasshopper control on nesting killdeer *(Charadrius vociferus).* M.S. thesis. Fort Collins, CO: Colorado State University. 41 p. + appendices.

Fair, J. M.; Kennedy, P. L.; McEwen, L. C. 1995. Effects of carbaryl grasshopper control on nesting killdeer in North Dakota. Environmental Toxicology and Chemistry 14: 881–890.

Frocke, J. B. 1983. The role of nestboxes in bird research and management. In: Snag habitat management: proceedings of the symposium. Gen. Tech. Rep. RM-99. Ft. Collins, CO: U.S. Department of Agriculture, Forest Service, Rocky Mountain Forest and Range Experiment Station: 10–13.

George, T. L.; McEwen, L. C.; Fowler, A. 1992. Effects of a carbaryl bait treatment on nontarget wildlife. Environmental Entomology 21: 1239–1247.

Grue, C. E.; Powell, G.V.N.; McChesney, M. J. 1982. Care of nestlings by wild starlings exposed to an organophosphate pesticide. Journal of Applied Ecology 19: 327–335.

Grue, C. E.; Fleming, W. J.; Busby, D. G.; Hill, E. F. 1983. Assessing hazards of organophosphate pesticides to wildlife. Transactions of North American Wildlife and Natural Resources Conference 48: 200–220.

Heinz, G. H. 1989. How lethal are sublethal effects? Environmental Toxicology and Chemistry 8: 463–464.

Heinz, G. H.; Hill, E. F.; Stickel, W. H.; Stickel, L. F. 1979. Environmental contaminant studies by the Patuxent wildlife research center. In: Kenega, E. E., ed. Avian and mammalian wildlife toxicology. Spec. Tech. Publ. 693. Philadelphia: American Society for Testing and Materials: 9–35.

Henny, C. J.; Blus, L. J.; Stafford, C. J. 1983. Effects of heptachlor on American kestrels in the Columbia Basin, Oregon. Journal of Wildlife Management 47: 1080–1087.

Johnson, R. R.; Haight, L. T.; Riffey, M. M.; Simpson, J. M. 1980. Brushland/steppe bird populations. In: Workshop proceedings: management of western forests and grasslands for nongame birds. Gen. Tech. Rep. INT-86. Ogden, UT: U.S. Department of Agriculture, Forest Service, Intermountain Forest and Range Experiment Station: 98–113.

Kirk, D. A.; Evenden, M. D.; Mineau, P. 1996. Past and current attempts to evaluate the role of birds as predators of insect pests in temperate agriculture. Current Ornithology 13: 175–269.

Kreitzer, J. F.; Spann, J. W. 1968. Mortality among bobwhites confined to a heptachlor contaminated environment. Journal of Wildlife Management 32: 874–878.

Loftin, R. W. 1992. Use of nest boxes by the Florida kestrel. Florida Field Naturalist 20: 57–88.

Lower, W. R.; Kendall, R. J. 1990. Sentinel species and sentinel bioassay. In: McCarthy, J. F.; Shugart, L. R., eds. Biomarkers of environmental contamination. Boca Raton, FL: CRC Press: 309–332.

McEwen, L. C. 1987. Function of insectivorous birds in a shortgrass IPM system. In: Capinera, J. L., ed. Integrated pest management on rangeland: a shortgrass prairie perspective. Boulder, CO: Westview Press: 324–333.

Rattner, B. A.; Franson, J. C. 1984. Methyl parathion and fenvalevate toxicity in American kestrels: acute physiological responses and effects of cold. Canadian Journal of Physiology and Pharmacology 62: 787–792.

Smith, G. J. 1987. Pesticide use and toxicology in relation to wildlife: organophosphorous and carbamate compounds. Resour. Publ. 170. Washington, DC: U.S. Department of the Interior, U.S. Fish and Wildlife Service. 171 p.

Stickel, W. H. 1975. Some effects of pollutants in terrestrial ecosystems. In: McIntyre, A. D.; Mills, C. F., eds. Ecological toxicology research. New York: Plenum Publishing Corp.: 25–74.

Taira, T. 1994. Blood analysis of American kestrel and golden eagle nestlings exposed to malathion or carbaryl. M.S. thesis. Ft. Collins, CO: Colorado State University. 80 p.

U.S. Department of Agriculture, Animal and Plant Health Inspection Service. 1987. Rangeland Grasshopper Cooperative Management Program: final environmental impact statement. Washington, DC: U.S. Department of Agriculture, Animal and Plant Health Inspection Service. 221 p.

Wakeland, C. 1958. The high plains grasshopper. Tech. Bull. 1167. Washington, DC: U.S. Department of Agriculture, Agricultural Research Service. 168 p.

Wiemeyer, S. N.; Lincer, J. L. 1987. The use of kestrels in toxicology. In: Bird, D. M.; Bowman, R., eds. The ancestral kestrel. Raptor Res. Rep. 6. Ste. Anne de Bellevue, PQ: Raptor Research Foundation, Inc., and Macdonald Raptor Research Centre of McGill University: 165–178.

Wiemeyer, S. N.; Sparling, D. W. 1991. Acute toxicity of four anticholinesterase insecticides to American kestrels, eastern screech-owls and northern bobwhites. Environmental Toxicology and Chemistry 10: 1139–1148.

Wiens, J. A.; Dyer, M. I. 1975. Rangeland avifaunas: their composition, energetics, and role in the ecosystem. In: Smith, D. R., coord. Proceedings of the symposium on management of forest and range habitats for nongame birds; 6–9 May 1975, Tucson, AZ. Gen. Tech. Rep. WO-1. Washington, D.C.: U.S. Department of Agriculture, Forest Service: 146–182.